of 106,000 medical admissions to the wards and dispensary of the Johns Hopkins Hospital, two were due to hemochromatosis. Of thirty-five cases in the literature, including the two here reported, all but two were in males. With one exception the ages of the patient ranged from thirty to sixty years.

Hemochromatosis in its final stages presents a triad of symptoms: (1) Pigmentation of the viscera and, in the vast majority of cases, also of the skin; (2) hypertrophic cirrhosis of the liver—very occasionally atrophic cirrhosis; and (3) diabetes mellitus, a terminal feature, due to a sclerosis of the pancreas causing destruction of the islands of Langerhans and consequent interference with the secretion of the normal glycolytic ferment.

Death usually occurs within a year after the onset of the diabetic symptoms, although it was delayed till twenty months in Case I, herewith reported.

The balance of opinion is in favor of the view that hemochromatosis is due to some toxic agent of unknown character and origin, which results in an increased destruction of the red blood cells with deposition of hemosiderin and hemofuscin in the body tissues. Whether the changes in the liver and pancreas are due to the deposition of the pigment or to the same toxic agent which is supposed to cause the blood destruction is still a disputed question. It has been suggested that the excessive amount of iron in the tissues may be due to defective elimination rather than to increased formation from the blood.

ACUTE PULMONARY ŒDEMA, WITH SPECIAL REFERENCE TO A RECURRENT FORM.[1]

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The œdema of the lungs to which I desire to call attention is not that so commonly seen in the period of lost compensation of valvular or muscular heart disease, in cachectic states, in grave infectious diseases, or in acute and chronic nephritis in the terminal phase. It bears no relation to subcutaneous dropsy, and in typical cases is not associated with it. In the suddenness of its onset, in the alarming character of its symptoms, and in the mortal peril in which it places the patient, acute pulmonary œdema resembles angina pectoris, and deserves, with equal propriety, a separate place in our text-books. Those who, interested in the subject, look through the text-books and encyclopedias of medicine will be astonished to find how little mention is made of acute œdema of the lungs or, for that matter, of pulmonary œdema in general.

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[1] Read in abstract at the meeting of the Association of American Physicians, Washington, D. C., May 15 and 16, 1906.

HISTORY. Laënnec,[2] in 1819, alluded in his chapter on œdema of the lungs to a suffocative orthopnœa that sometimes carries off children after an attack of measles, and considered it an idiopathic anasarca of the lungs.

Andral,[3] in his commentary to the fourth edition of Laënnec's *Traité de l'Auscultation médiate,* clearly describes acute pulmonary œdema, and compares it with œdema of the glottis; but neither in this commentary nor in his own *Cours de Pathologie interne*[4] does he speak of the causes. He recognizes three forms of œdema: (1) A fulminant form (forme suraigue), in which the patient is suddenly seized with an intense dyspnœa that in a short time causes death; (2) a subacute form, in which the dyspnœa, though sudden in onset, lasts somewhat longer before death ensues; and (3) chronic œdema, which does not concern us at the present time.

Legendre,[5] in 1846, speaks of an acute pulmonary œdema occurring during the course of scarlet fever.

Rokitansky,[6] like Andral, compares acute pulmonary œdema with œdema of the glottis; it is, he states, a frequent cause of suffocation in adults and newborn children, and is often combined with hyperemia and serous effusion within the cavity of the cranium.

Bernheim,[7] in his clinical lectures, refers to a fulminant pulmonary œdema during the course of acute articular rheumatism; and Lebreton,[8] among the pulmonary complications of rheumatism, mentions an acute œdema of the type under consideration.

Landouzy[9] observed two rheumatic patients with pulmonary œdema, one having four and the other five recurrent attacks; neither had albuminuria.

Jaccoud[10] reports a case of typhoid fever terminating fatally within fifteen minutes from acute pulmonary œdema, which he designates as "un veritable coup de sang pulmonaire." The autopsy revealed nothing to explain the tragic event.

Debove[11] speaks of repeated accesses of acute pulmonary œdema, and cites the case of a physician having aortic disease who died in half an hour in an attack, after having had several before. Debove thinks that aortic affections, and cardiac diseases in general, predispose to acute pulmonary œdema.

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[2] Treatise on Diseases of the Chest and on Mediate Auscultation, 1830, p. 179.

[3] Laënnec's Traité de l'Auscultation médiate, 4ième édition, par Andral, t. xi, p. 424.

[4] Cours de Pathologie interne; 4ième édition, 1842, p. 123.

[5] Œdeme aigue du tissue cellulaire de poumon à la suite de la scarlatine, quoted by Fouineau.

[6] Manual of Pathological Anatomy, Sydenham Society's translation, iv, p. 71.

[7] Leçons de Clinique médicale, 1877, p. 494, quoted by Fouineau, q. v.

[8] Thèse de Paris, 1884, quoted by Fouineau.

[9] Bulletin médical, 1897, p. 445.

[10] Clinique de la Pitié, i, 1883, 1884, p. 567.

[11] Bulletin médical, 1897, xi, p. 400.

P. de La Harpe,[12] in 1881, reported three cases that illustrate the condition of acute pulmonary œdema very well: Case I. A woman, aged forty-three years, healthy, exposed herself to inclement weather after a slight attack of influenza; œdema of the lungs developed and caused her death within a short time. Case II. A woman, aged sixty years, also healthy, after one or two nocturnal attacks, died of œdema in a few minutes. Case III. A man had from fifteen to twenty attacks, all terminating by repeated eructations of gas and the voiding of large amounts of almost colorless urine. Similar cases are reported by Lund.[13]

One of the most important communications is that of Dr. Lindlay Steven,[14] of Glasgow, who, under the name of acute suffocative pulmonary œdema, reports two cases, in both of which there were evidences of renal disease; and in one of them there was also an affection of the aortic valve. Steven's paper stimulated the report of a number of other cases, one of the most interesting being that of Lissaman,[15] whose patient, a woman, aged forty-five years, had within two and a half years seventy-two attacks, all characterized by sudden onset, pain in the chest, frightful dyspnœa, lividity of the face, profuse cold sweats, labored action of the heart with small and rapid pulse, and the ejection, without cough, of enormous quantities of thin, pink, seromucous fluid. All the attacks were nocturnal.

Four well-studied cases have been reported by Hewlett,[16] who adopts Steven's designation of acute suffocative pulmonary œdema: Case I. A woman, aged forty-nine years, had six attacks—two in the afternoon, the others at night. Albuminuria, myocarditis, arteriosclerosis, and hypertension. Case II. A woman, aged fifty-two years, had repeated attacks, after each of which albumin and casts were present in the urine. In the intervals only casts were found. Heart hypertrophied; blood-pressure high. Case III. A woman, aged sixty-nine years, had ten attacks, all but one at night. Urine contained a trace of albumin; no casts. Case IV. An alcoholic woman, aged fifty-two years, who died in an attack of œdema.

A fairly typical case of the recurrent variety is reported in this country by Crummer.[17]

Among the most important systematic contributions to the subject from the clinical and pathological standpoint should be mentioned those of Huchard,[18] Dieulafoy,[19] Renaut,[20] Fuoineau,[21] Bouveret,[22] and Coplin.[23]

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[12] Rev. méd. de la Suisse Rom., 1881, p. 322.

[13] Lund. Norsk. Mag. for Lägevid, quoted by Huchard and by Fouineau.

[14] Lancet, January 11, 1902.

[15] Ibid., February 8, 1902.

[16] Intercolonial Medical Journal, 1903, p. 611.

[17] Northwestern Lancet, 1902, p. 203.

Its pathogenesis has been studied by Welch,[24] whose researches are of fundamental importance; by Sahli,[25] Grossman,[26] Löwit,[27] and others.

ETIOLOGY. Acute pulmonary œdema is associated with a variety of affections that, in a clinical sense, may be considered in the light of causes. I would classify them as follows: (1) Arteriosclerosis; (2) Bright's disease; (3) heart disease—angina pectoris; myocarditis; valvular affections; (4) asthma; (5) acute infectious diseases—typhoid fever, measles, rheumatic fever, influenza, pneumonia, etc.; (6) pregnancy; (7) paracentesis of thorax and of abdomen; (8) angioneurotic œdema; and (9) obscure conditions of questionable causal relationship (hysteria, etc.).

1. *Arteriosclerosis.* A large number of the reported cases of acute pulmonary œdema showed arterial disease. Case II of my series belongs to this group. Huchard[28] contends that vascular disease, especially aortitis and periaortitis, is the only cause of acute pulmonary œdema. No doubt it is an important factor in many cases, but not the essential one in all. Clifford Allbutt[29] admits the importance of aortitis; but very properly lays stress on the coexistence of general arteriosclerosis. The œdema results, according to Huchard, from disturbance in the innervation of the pulmonary vessels brought about by extension of the aortitic or periaortitic process to the cardiopulmonary plexus.

2. *Bright's Disease.* The important relationship that Bright's disease bears to acute pulmonary œdema was first clearly pointed out in 1890 by Bouveret, and afterward emphasized by Dieulafoy, Brouardel,[30] and others. Case I of my series is an illustration.

Dieulafoy, like Andral and Rokitansky, compares œdema of the lungs to œdema of the glottis, which is known to be one of the occasional complications of Bright's disease. How the nephritis acts has been much discussed by French writers: whether directly by toxemia (uremic poisoning) or indirectly through disease of the heart and aorta. For myself, I believe that nephritis can produce acute pulmonary œdema without the intervention of arterial or cardiac disease, through the mediation of poisons, the true nature of which remains unknown.

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[18] Bulletin médicale, 1897, xi, p. 393.

[19] Bulletin médicale, 1897, xi, p. 400.

[20] Bull. de l'Acad. de Méd., 1897, xxxvii, p. 557.

[21] L'Oedème du Poumon, Thèse de Paris, 1898.

[22] Rev. de méd., March, 1890.

[23] Therapeutic Gazette, April, 1906.

[24] Arch. f. path. Anat. und Phys., 1878, lxxii, p. 375.

[25] Arch. f. exper. Path. und Pharm., 1885, xix, p. 435.

[26] Zeit. f. klin. Med., xii, xv, vxi, 1887–1890.

[27] Ziegler's Beiträge, xiv, p. 401; Centralblatt f. allgem. Path. und pathol. Anat., 1895.

[28] Loc. cit.

[29] Lancet, July 8, 1903, p. 141.

[30] Bull. de l'Acad. de Méd., 1897, xxxvii, p. 509; Bulletin médicale, 1897, xi, p. 400.

3. *Heart Disease.* I have separated this from arteriosclerosis, with which it is generally associated, because there are cases of œdema in the literature in which the last condition is not mentioned, and may be presumed not to have existed, but in which there was valvular disease of the heart. It must be remembered that the œdema here considered is to be distinguished, as I stated in the beginning of the paper, from the ordinary œdema of valvular disease, which is a part of a general œdematous process.

With regard to angina pectoris, the coincident occurrence of œdema is noted but by few authors. It is presented in Case V of my series. Angina pectoris is, no doubt, often associated with aortitis, but not necessarily so; and certainly not always with evident peripheral arterial disease. In my own case there was no general arteriosclerosis. I have for that reason separated the pulmonary œdema of angina pectoris from that of arteriosclerosis.

4. *Asthma.* The occurrence of acute suffocative œdema during the course of asthma is illustrated by two of my cases—III and IV. Differentiation of the condition from a violent asthmatic paroxysm can be made by attention to the physical signs, the character of the expectoration, and the profound collapse. Moreover, the patients themselves are aware that something very different from an asthmatic attack is taking place. It is quite probable that many of the so-called asthmatic attacks coming on in middle or advanced life, without any previous history of asthma, are in reality attacks of pulmonary œdema.

5. *Acute Infectious Diseases.* In the section on history I have already cited a number of authors who have observed acute pulmonary œdema during (*a*) typhoid fever (Jaccoud and Merklen[31]); (*b*) rheumatic fever (Lebreton and Landouzy); (*c*) scarlet fever (Legendre); (*d*) influenza (Glasgow[32] and Lemoine[33]); (*e*) pneumonia (Souin de la Savinière[34]).

6. *Pregnancy.* The first to report a case of acute pulmonary œdema during pregnancy was Charles Wilson[35] (1856), whose patient was a woman, aged thirty-four years, eight months pregnant. There was anasarca and scanty, high-colored urine (no analysis); sudden pulmonary œdema at night; recovery after abstraction of twenty ounces of blood. Premature, spontaneous labor, followed by puerperal mania, with recovery under opium.

Vinay,[36] in 1896, reported three cases of acute pulmonary œdema during pregnancy, two of which were speedily fatal; in all three there was mitral-valve disease, and in one of them also nephritis. One of my cases, VI, belongs to this group.

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[31] La Méd. moderne, 1903, xiv, p. 145.

[32] Amer. Jour. Med. Sci., April, 1895, p. 425.

[33] Press méd., 1905, p. 151.

[34] Contribution à l'etude de l'œdème aigue du poumon, Thèse de Paris, 1873, quoted by Fouineau.

[35] Edinburgh Med. Jour., 1856, ii, part i, p. 481.

[36] Lyon méd., 1896, lxxxii, p. 289.

7. *Paracentesis of the Thorax and of the Abdomen.* In rare instances tapping of the chest, and more rarely still of the abdomen, is followed by the signs of acute pulmonary œdema, with the expectoration of large amounts of frothy albuminous material (albuminous expectoration). Originally it was believed that the albuminous expectoration was due to a wound of the lung, with a sucking up and the expectoration of the pleural fluid. This is not the case. The œdema results from an afflux of blood to the expanding lung, and a leakage of serum through the walls of the vessels, which by reason of long compression have become changed and rendered more permeable. In a paper upon this subject,[37] in which a bibliography will be found, I speak of this afflux as "congestion by recoil."

8. *Angioneurotic Œdema.* A case of sudden pulmonary œdema with coincident fugitive swelling of the face is reported by Hermann Müller,[38] who looks upon the pulmonary œdema as of the same origin, namely, angioneurotic, as of that of the face.

9. *Obscure Conditions of Questionable Causal Relationship.* Lavallée[39] reports a case of tabes dorsalis with two attacks of localized pulmonary œdema. There was arteriosclerosis, but no albuminuria and no valvular disease. In a case reported by Léopold-Lévy,[40] there was no other morbid condition than hysteria. Poppert[41] observed acute œdema of the lungs after ether anesthesia and collected seven similar cases from the literature.

Pathogenesis. Pulmonary œdema, whether acute or chronic, is brought about by leakage of serum through the capillary walls into the air vesicles and intervesicular tissues. In order that such leakage may take place, the pressure within the capillaries must be raised, or the permeability of the walls must be increased, or both factors must co-exist.

Many are the theories that have been advanced to explain the acute form of œdema. For purposes of clearness they may be classified as follows: (1) The periaortitic theory; (2) the cardiac theory; (3) the toxic vasomotor theory; (4) the infectious theory; and (5) the increased permeability theory.

1. *The Periaortitic Theory.* Huchard believes that the principal cause of acute pulmonary œdema is aortitis or periaortitis with extension of the inflammatory process to the cardiopulmonary plexus. The pathogenetic sequence, to quote his words, is as follows: "Disturbances of the innervation of the cardiopulmonary plexus by periaortitis; considerable augmentation of the vascular tension in the small circulation; acute or rapid insufficiency of the right ventricle." This is a satisfactory theory for those cases in which angina pectoris, Hodgson's disease, or other affections of the

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[37] Amer. Jour. Med. Sci., April, 1902.

[38] Corresp. f. Schweizer Aerzte, 1891, p. 432.

[39] Journ. des Praticiens, May 14, 1897.

[40] Arch. de méd., 1895, clxxvi, p. 473.

[41] Deut. med. Woch., 1894, p. 719.

aorta exist; but it does not give an adequate explanation of those cases in which evidence of aortitis and general arterial disease is absent.

2. *The Cardiac Theory.* The cardiac origin of pulmonary œdema has its ablest defender in Welch, whose classical essay from the laboratory of Cohnheim has been misunderstood by more than one later investigator. As restated by Welch[42] in a recent communication, the theory is as follows: "Pulmonary œdema is due to a disproportion between the working power of the left ventricle of such character that, the resistance remaining the same, the left heart is unable to expel in a unit of time the same quantity of blood as the right heart. Such disproportion may be brought about by paralysis or by spasm of the left ventricle." The salient feature of this theory then is a difference in the amount of blood thrown out by the two ventricles; the left expelling a little less in a given time than the right. As a result some blood remains behind in the left auricle; this reacts upon the pulmonary veins, so that they cannot empty themselves completely; consequently, there is a rise of pressure in the pulmonary circulation to a degree sufficient to bring about a transudation of fluid through the capillary walls.

The attacks upon this theory by Grossmann, who has produced pulmonary œdema with muscarin, by Sahli, and by Löwit are answered by Welch in the communication referred to, and need not be taken up here. Meltzer[43] has ingeniously applied Welch's theory to an explanation of the pulmonary œdema following adrenalin injection. He holds that in consequence of the great obstacle caused by the considerable constriction of all the smaller blood-vessels of the body, the left ventricle is incapable of throwing out all the blood that it receives from the pulmonary veins, while, on the other hand, the right ventricle, stimulated by adrenalin, unloads with increased energy upon the lungs all the blood which the contracting vessels drive into it.

3. *The Toxic Vasomotor Theory.* According to this theory, which is advanced by Bouveret,[44] acute pulmonary œdema is due to the action of toxic substances on the pulmonary vasomotor mechanism. There is doubt among physiologists whether the pulmonary arteries possess a vasomotor nerve supply, although Francois-Franck[45] seems to have shown that such an one does exist, and Hofmann[46] considers it practically demonstrated that the pulmonary vessels have vasomotor nerves, though their influence is not great.

The toxic theory is useful in explaining the acute pulmonary œdema of some cases of Bright's disease, that of angioneurotic œdema, and, perhaps, that of pregnancy when there is no valvular or vascular disease.

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[42] Meltzer, Amer. Med., 1904, viii, p. 19, 59, 151, 191.

[43] Loc. cit.

[44] Loc. cit.

[45] Arch. de Physiol., 1895, p. 816, quoted by Hofmann in Handbuch der Physiol. des Menschen, Nagel, p. 298.

[46] *Handbuch der Physiol. des Menschen*, Nagel, p. 300.

4. *The Infectious Theory.* Caussade, Milhit, and Israel de Jong[47] have recently described a subacute œdema accompanying diverse pulmonary affections and possessing a more or less epidemic character. The expectoration is albuminous, frothy, not sanguinolent, and is easily differentiated from that peculiar to the disease, *e. g.*, bronchitis or pneumonia, with which the œdema happens to be associated. In the sputum the pneumococcus, usually of a virulent type, is present; whence the authors conclude that it is the cause of the œdema. Chloride retention and the neuroarthritic diathesis are predisposing factors.

The mere presence of the pneumococcus hardly possesses the importance the authors give to it, for such presence is to be expected when the œdema is an accompaniment of inflammations of the lungs that are known to be associated with this organism. The possibility of an infectious pulmonary œdema is, however, undeniable. It is probably present at some stage of all acute inflammations of the lung; but the type of œdema under consideration here does not bear the stamp of an infectious origin. The sudden onset of the attacks—their brevity, their subfebrile or afebrile character—militate against such a view.

5. *The Increased Permeability Theory.* Increased permeability as the sole cause of acute pulmonary œdema, as advocated, among others, by Martin,[48] is not adequate; but in conjunction with increased blood pressure it becomes an important factor, especially in the form of pulmonary œdema following paracentesis of the thorax.

PATHOLOGY. Renaut[49]—who is of opinion that both anatomically and clinically acute pulmonary œdema possesses an individuality that indicates a special pathogenesis—finds that the principal lesion is a serous inundation of the pulmonary alveoli. The alveolar fluid may be slightly fibrinous. White cells are abundant, sometimes to such an extent as to resemble a pneumonic exudate. That the entrance of the liquid into the alveoli has occurred violently under pressure is shown by the many points of rupture in the alveolar walls. The epithelium disappears within the limits of the œdema, having been forced off by the sudden eruption of fluid. The capillaries of the alveolar walls are flattened and rendered impermeable by compression, and, hence, are devoid of red cells. The large pulmonary and bronchial veins are, however, engorged with blood. The lymphatic spaces under the pleura are also distended.

Coplin,[50] whose study is the most recent, finds a finely granular deposit in the distended air vesicles and interstitial tissue, representing an albuminous fluid coagulated by the fixative. The capillaries usually show no conspicuous overdistention, and are frequently empty. The septal walls are often loosened and their tissues dissociated and infiltrated with mononuclear cells. The cell content of the air vesicles and interstitial tissue is subject to considerable variation; in some cases the erythrocytes are plentiful, and in others, not; polymorphonuclear leukocytes are at times abundant, and then bacteria are also present. In other cases again the cells are of the mononuclear type. Unlike Renaut, Coplin holds that detached vesicular epithelium can always be demonstrated. In some cases there is a marked swelling of the submucosa of the smaller bronchi and bronchioles, with a consequent narrowing of the lumen. The elastica is often stretched and the subserous and interstitial lymph vessels are distended.

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[47] Bull. et mém. de la Soc. méd. des Hôp. de Paris, February, 1906, p. 160.

[48] See Hewlett, Intercolonial Medical Journal, 1903, p. 611.

[49] Loc. cit.

[50] Loc. cit

SYMPTOMATOLOGY. One of the most striking features of acute pulmonary œdema is the suddenness of its onset, often in what appears to be a state of good health. The patient is seized, usually at night, with a sensation of oppression and suffocation accompanied by a fear of dissolution. The breathing is labored and of a rattling character; the dyspnœa, or orthopnea, intense; the countenance either livid or pale and bedewed with sweat; cough may be harassing, or it may be entirely absent. In typical cases there is a copious, frothy, albuminous expectoration which may be expelled in gushes from the mouth and nose, and may reach the amount of one or two liters during an attack. Often it is blood-tinged, and in one of my cases (II) it had the appearance of raspberry vinegar; sometimes it is whitish like bronchial secretion. The action of the heart and the pulse are feeble in some cases, as in the two with asthma here reported; the pulse, however, may be full and bounding. Over the lungs the characteristic subcrepitant and bubbling rales of œdema are heard; not rarely, they are most abundant over the upper parts, while in the ordinary œdema they are most numerous over the lower lobes posteriorly.

The first attack of œdema may be fatal, like the first attack of angina pectoris. More frequently the first attack is not fatal, and is followed by numerous others, all presenting a striking uniformity and each placing the patient in imminent danger of death. For some reason the paroxysms are generally nocturnal, and not rarely they set in during sleep. The average duration varies from three to six hours; some cases last twelve to twenty-four hours; in the extremely fulminant types, as in Case I of my series, death may take place within ten or fifteen minutes, the patient literally drowning in his own juices.

Brouardel[51] has pointed out the medicolegal importance of these hyperacute cases in which sometimes there is no expectoration, death taking place by suffocation before the patient has a chance to rid himself of any fluid. As to the number of attacks, the case of Lissaman[52] with seventy-two seems to hold the record. It is important to bear in mind this tendency to recurrence, so as to give a reasonable prognosis, and to be prepared to treat the attacks on a definite, well-considered plan.

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[51] Loc. cit.

Cerebral symptoms may be present, as in Case II, in which aphasia and hemiplegia complicated the attack and outlasted it for some time. Delirium, convulsions, and coma have also been noted.

DIAGNOSIS. *Acute pulmonary œdema must be distinguished* from (*a*) asthma; (*b*) pulmonary embolism; (*c*) acute dilatation of the heart; and (*d*) uremic asthma.

Asthma. From asthma, acute pulmonary œdema may be distinguished by the fact that in the former there is very little expectoration during the attack, and is established only when the seizure has passed off, and is limited to a small amount of glairy material or a few mucous pellets. The piping rales of asthma are different from the mucous, bubbling rales of œdema.

Pulmonary Embolism. In pulmonary embolism there is an embolic source; expectoration is developed later than in acute pulmonary œdema, in which it usually coincides with the onset of dyspnœa; when once established in embolism it is less abundant and more definitely bloody than in pulmonary œdema.

Acute Dilatation of the Heart. The dyspnœa and cyanosis in acute cardiac dilatation are rarely as intense as in acute pulmonary œdema. There may be œdema of the lungs; the difference here is perhaps only one of degree. In typical cases of acute pulmonary œdema the dominant feature is the inundation of the lung with fluid, and the dyspnœa is more directly mechanical than in dilatation of the heart.

Uremic Asthma. The dyspnœa in uremic asthma is seldom as severe as in acute pulmonary œdema, nor is it accompanied by profuse frothy expectoration, and, hence, not by the characteristic physical signs of œdema of the lungs.

TREATMENT. Treatment must be prompt to be effective. The best results are obtained by venesection. Merklen,[53] with great enthusiasm, says, "c'est l'unique salut." The abstraction of a small amount of blood usually suffices, though in rare instances as much as from 400 to 600 c.c. has to be taken. When bleeding cannot be done, or in addition thereto, dry cups may be used. Half a dozen wineglasses, in which air has been rarefied by burning alcohol, should be placed over the back; and if the patient is turned on his side several can be put on the front of the chest at the same time. If there is much nervous excitement, and a terrifying feeling of impending death, a small dose of morphine should be administered hypodermically. Authors, it must be admitted, differ as to the value of morphine. Hewlett recommends it; Steven condemns it. Lissaman found chloroform eminently successful in his remarkable case. When the pulse is feeble and asystole is threatened, diffusible stimulants, such as whiskey, ammonia, and hypodermic injections of camphor, are useful. Crummer ascribes the favorable outcome in his case to adrenalin chloride; but as far as this drug is concerned, the experimental observations of Bouchard and Claude,[54] of Meltzer, and of Coplin, who found that it could produce œdema in the lower animals, ought to make us cautious. Nitroglycerin suggests itself as a remedy in those cases in which there is every evidence of arterial hypertension, namely, in the arteriosclerotic and renal types. Oxygen is an agent of value when there is much cyanosis.

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[52] Loc. cit.

[53] Loc. cit.

As I have said before, the likelihood of recurrence should be borne in mind, and should be made clear to the relatives of the patient. Where possible an intelligent member should be instructed in the method of applying dry cups, and, perhaps, in the use of the hypodermic syringe. Such precautions commend themselves more especially because the attacks generally occur at night, when it may be difficult to obtain medical aid at once.

REPORT OF CASES: CASE I.—Mrs. L., aged forty-six years; numerous attacks of acute œdema coming on without apparent cause. First attack in 1902, two days after death of mother; lasted one and one-half hours. No albuminuria in the beginning, later small amount of albumin, urine pale, low specific gravity, casts from time to time. Attacks characterized by dyspnœa, rattling breathing, mucous rales, sense of fear and terror, high tension pulse. Patient neurotic and subject to laughing and crying spells. Death occurred, December, 1905, in an attack lasting ten minutes, during which frothy, bloody fluid gushed from mouth and nose. Autopsy: Chronic interstitial nephritis, hypertrophy of the heart; intense pulmonary œdema, fresh petechial hemorrhages in the pleura and pericardium.

CASE II.—Mr. W., aged seventy-two years; always healthy. Seized suddenly one night with intense œdema of the lungs; chest filled with moist rales; reddish, frothy fluid gushing from mouth; dyspnœa, collapse, unconsciousness, paralysis of right arm and leg, pulse irregular, reduplication of first sound at apex. Marked arteriosclerosis, urine (catheter) albuminous. Disappearance of pulmonary signs in two or three days, palsy lasting a little longer; eventual recovery. Another attack a month later, January 23, 1904, not so severe; recovery. Numerous others, usually with stupor or coma and variable degrees of fleeting palsy and aphasia. Last attack, March 19, 1904, at 3 A.M.; intense dyspnœa, unconsciousness, fluid gushing from mouth and nose, paralysis of right arm; partial restoration of consciousness following venesection. Death, no autopsy.

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[54] Comp. rend. de l'Acad. des Sciences, December, 1902, p. 928.

CASE III.—Mrs. McC., aged forty years; asthma of several years' standing. During the past winter and spring repeated attacks of intense dyspnœa with rattling breathing, frothy expectoration, bubbling rales, alarming collapse, with pallor, feeble pulse, clammy skin. Attacks entirely different from asthmatic paroxysms, but occurring during asthmatic periods. A number of attacks within two or three months. None since going south.

CASE IV.—Miss I. C., aged twenty-five years; asthmatic. Without apparent cause seized at midnight with intense dyspnœa and fear of impending death; lips cyanotic, face ashen pale, skin clammy, no pulse at wrist, respiration rattling, chest filled with bubbling rales, frothy fluid slightly tinged with blood gushing from mouth and nose. Urine normal. Attack subsided by morning; extreme prostration for several days; one slight recurrence while still in bed.

CASE V.—Mrs. N. S., widow, aged fifty-eight years. For three or four years attacks of anginoid pain in precordium with radiation into left arm. Attacks always associated with orthopnœa and signs of pulmonary œdema; the latter at times confined to left lung, generally bilateral. Best results during attacks from dry cupping and nitroglycerin. Death from cardiac dropsy, no autopsy.

CASE VI.—Mrs. S., aged thirty-eight years; pregnant seven months —eighth pregnancy. Seized without warning and without previous exposure with an attack of intense dyspnœa and a feeling of oppression and anxiety. There was a great deal of wheezing and cyanosis, harassing cough, and alarming prostration. My first impression was that the patient had asthma; but antiasthmatic treatment had no result. The rales, which had been dry, soon became moist and bubbling, and a free, whitish, frothy expectoration developed. The patient had never had asthma nor any other illness; had passed through five labors and three miscarriages without a doctor, a midwife only attending. There was no history of previous kidney disease; nevertheless, the urine was loaded with albumin, but free from casts. The heart presented nothing abnormal; there was no arteriosclerosis. Her condition becoming worse, and as she seemed in danger of suffocation, Dr. E. E. Montgomery was called in consultation, and it was agreed between us that labor should be promptly induced. Under ethyl-chloride anesthesia an attempt was made to stretch the mouth of the womb, but it could not be done sufficiently, and it was thought best to introduce a piece of gauze and await developments. Free bleeding occurred during the stretching. To our surprise labor did not follow, but the patient rapidly improved, and within twenty-four hours had practically recovered from her dangerous condition. The urine cleared up entirely within a few days. Six weeks later, without any warning, the œdema returned. I promptly bled her from the elbow, with the most happy result. Her confinement is expected at any day.[55]

CONCLUSIONS. 1. There is an acute pulmonary œdema that comes on without any apparent exciting cause and often kills in a short time.

2. Recovery from an attack is frequent; but there is a striking tendency to recurrence, subsequent attacks being similar to the first. They set in suddenly, usually at night, and place the patient's life in jeopardy.

3. The clinical causes are various: arteriosclerosis and cardiac and renal disease are most important.

4. The pathogenesis is obscure; vasomotor disturbances and a disproportionate activity of the two ventricles (the right predominating) are the chief factors.

5. The chief symptoms are agonizing dyspnœa, cyanosis, cough, expectoration of frothy albuminous fluid, and profound prostration. Over the lungs, often more over the upper than the lower portions, the characteristic moist rales of edema are heard.

6. The most valuable remedies are bleeding, dry cupping, and cardiac stimulants.

7. The condition is of sufficient importance to warrant its having a special chapter in the text-books of medicine.

THE NATURE OF APLASTIC ANEMIA AND ITS RELATION TO OTHER ANEMIAS.[1]

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(From the William Pepper Laboratory of Clinical Medicine, Phoebe A. Hearst Foundation.)

APLASTIC anemia was first described by Ehrlich[2] in 1888. Since that time isolated cases have been reported and recently the condition has received considerable attention. The following typical case occurred in the University Hospital in the service of Dr. Stengel, to whom I am indebted for the clinical data.

J. C., female, aged thirty-three years, white, married, was admitted

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[55] Since writing this she was delivered without any difficulty by Dr. V. A. Loeb, to whom I transferred her for this purpose; she has had no other attacks.

[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 15 and 16, 1906.

[2] Charité Annalen, 1888, xiii.